Performance of PTSD Patients on Standard Tests of Memory

Implications for Trauma[a]

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Use of neuropsychological tests in posttraumatic stress disorder (PTSD) began approximately 10 years ago with the growing interest in reported concentration and memory problems in trauma survivors. Clinicians in particular were familiar with patients' descriptions of difficulties attending to and recalling basic factual data while concomitantly reporting distressing, intermittently recurrent intrusive memories of traumas. This seeming dichotomy in performance could be roughly characterized in two ways: (1) a nonspecific concentration or memory deficit in this disorder, and (2) the tendency to selectively attend to or encode cues related to traumatic experiences. These two approaches had differing implications: a focus on memory problems in general could suggest a more basic cognitive disturbance previously described in other anxiety and depressive disorders, with limited specificity for PTSD, or an alteration in learning or processing capabilities that might be distinctive for trauma syndromes.[1,2]

This paper provides an overview of the topic of memory performance in PTSD, beginning with common etiological frameworks that have been employed to examine this issue. Next, extant use of neuropsychological tests, including standard and empirical measures, are reviewed followed by a summation of performance findings to date. The article concludes by reviewing the concordance between current models of memory disturbances and cognitive changes in PTSD and proposes a series of issues that need to be addressed in future research on this topic.

CONCEPTUAL MODELS

Basic Memory Models

The application of general theories of memory to purported learning deficits in PTSD is relatively broad at this time. Some models have suggested that recall and learning could be adversely affected by factors ranging from fluctuations in general

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attention or concentration to decreased motivation or affective condition of the respondent (e.g., depression).[3] These observations reflect mechanisms that have relevance for understanding the cognitive deficits found in other major psychiatric disorders (e.g., major depression and panic disorder). These models generally predict that memory problems in affective or anxiety disorders stem from processing deficits that are based in affective and sensory dysregulation.[4] Still, although PTSD shares certain phenomenological characteristics with these disorders (e.g., agitation and depressed mood), currently few, if any, empirical data exist to confirm any commonality in either core memory disturbances or underlying functional mechanisms.

More detailed probes into memory changes in PTSD have speculated that alterations in certain brain neurotransmitter systems (e.g., catecholamines) are likely to have an impact on memory and recall. Preclinical and clinical research in fact substantiate that noradrenaline and, recently, serotonin, are linked to basic changes in attention and concentration (e.g., disruptions in selective attention).[5] Furthermore, psychobiological research in PTSD confirms that central noradrenaline functions are abnormal in PTSD,[6] and preclinical research demonstrates that noradrenergic neurons in the thalamus and amygdala are selectively activated by stress. Since fear conditioning, a basic component of PTSD, is mediated through sensory pathways in subcortical brain systems that project to the thalamus and amygdala, it is not surprising that PTSD patients might evidence disruptions in cognitive processing, at least under conditions of high arousal or fear.[7] Thus, memory changes in this disorder could stem from the involvement of complex neural mechanisms and brain chemicals implicated in the response (or adaptation) to intense fear.[8]

Support for neurotransmitter relevance in cognition in PTSD is also found in the growing number of studies which show that PTSD is associated with significantly slowed reaction times on tasks involving the detection of target stimuli,[5] a finding that has been supported for both neutral[5] as well threat-based stimuli.[9–11] One possible explanation for these findings is that sustained or selective attentional disturbances affect the rate and depth of subsequent processing, rendering individuals vulnerable to interference, complex processing demands, and inefficient encoding. In this scenario, neurochemical dysregulation in PTSD is linked to memory problems largely through disturbances in attentional systems, the normal foundation for preserved recall.

Evidence of decreased hippocampal volume in some PTSD patients has recently suggested that actual neuroanatomic changes in the brain, possibly at the limbic or paralimbic level, might be linked to the development of cognitive changes.[12] Research by a number of investigators[13] has emphasized the importance of subcortical structures, such as the hippocampus, for certain components of memory (e.g., the explicit memory processing needed for memory consolidation). Accordingly, the permanency of memory traces could be impacted by structural changes in these brain regions. Still, despite preliminary data, empirical evidence that ties subtle neuroanatomical alterations to measurable cognitive change remains limited at this time[14] as does the specificity of such a finding for PTSD.[15]

Network Memory Models

In contrast to general models of memory, information-processing network models propose that memory changes or deficits in PTSD depend on the existence of closely

interlinked, semantic networks whose associational nodules are laid down during trauma.[16,17] These trauma-specific associations often generalize to other stimuli through secondary and higher order conditioning and are subsequently activated during the intense reexperiencing phenomena and phasic arousal found in PTSD. Recently, Metcalfe and Jacobs[18] suggested certain neuroanatomic correlates for network memory models in PTSD. Specifically, the hippocampus is seen as related to a "cool-cognition" dimension, while the amygdala is associated with a "hot-emotion" function. On the basis of this differentiation, the two brain structures are separately implicated in PTSD: traumatization, for example, substantially impacts the "cool" system by disabling certain of its functional cognitive capabilities, while the "hot" system associated with the amygdala becomes hyperresponsive. In actual neurologic disease, hippocampal disturbances would likely result in cognitive deficits in spatial perception, spatial memory, and episodic memory, while overall cognitive skills remain intact. In PTSD, where involvement of this brain region is likely to be more diffuse or nonspecific, cognitive abilities could remain intact, but fear-provoking stimuli would be liable to encoding in the absence of an adequate temporospatial context. Accordingly, many trauma memories could not be reliably retrieved from episodic memory. In contrast to the preceding, activation of the amygdala and associated structures could contribute to the phasic appearance of intense, intrusive thoughts and trauma memory fragments.

Overall, models like these help to emphasize the critical role played by stimuli with affective connotations, suggesting that PTSD is not necessarily linked to any clearcut storage or retrieval deficit per se but, rather, to disruptions at the intial encoding level, at least for certain types of information. Furthermore, these models offer some of the first evidence for bidirectionality in memory performance in PTSD; that is, under certain conditions, memory abilities could actually be enhanced, while under others, they are diminished.

Resource Allocation Models

Resource allocation models are similar to network models in that they propose the effects of an interlinked series of cognitive mechanisms in PTSD. Similar to cognitive and memory models for clinical depression, resource allocation theories suggest that the selective deployment of attentional skills to certain stimuli leads to their heightened accessibility. Under particular conditions, however (e.g., those involving competing task demands or complex, multilevel processing), the performance by a PTSD patient suffers appreciably because of the competition among fixed allocation resources. In this scenario, the level or complexity of a task could produce interference or disruption in required inhibition by drawing focused attention elsewhere. Consequently, the rapidity or accuracy of recall is likely to be seriously compromised. Research by McNally and colleagues[19] is particularly informative in this area. These investigators conducted experimental cognitive studies in PTSD and found growing evidence that certain characteristics of this disorder substantially disrupt correlates of normal information processing. As a result, some PTSD patients demonstrate measurable disturbances in normal attention and attendant recall when fear-based or trauma stimuli are used. Furthermore, the data suggest that cognitive

patterns in PTSD are notable for a pronounced bias towards high threat stimuli. Additional studies are required to determine whether baseline attention and memory are intact during more complex tasks in the context of these demonstrated aberrations at higher levels.

ASSESSMENT PARADIGMS

To assess memory in PTSD, clinicians and researchers have selected from a range of extant neuropsychological tests, with a primary interest on recall and effects of interference. Tests of recognition memory have generated somewhat greater interest, probably because of their less challenging nature in a population not known to have discrete brain lesions. Standard memory tests (e.g., Wechsler Memory Scale[20] and its revised version [WMS(-R)][21]) have been used to assess immediate and delayed verbal and nonverbal visual memory,[22] while more complex tasks (e.g., California Verbal Learning Test [CVLT][23] and Selective Reminding Test [SRT][24,25]) are employed when the study of memory correlates (e.g., retention capacity, efficiency, and interference effects) are of interest.

To date, studies using tests such as the WMS(-R) have yielded, at best, equivocal evidence for memory impairment in PTSD.[26] Overall, these and other data[27] suggest that most PTSD patients are not likely to have appreciable memory deficits *in the absence of* histories of diagnosed neurologic disease, head injury with loss of consciousness, or notable developmental (learning) disabilities, all factors that could impact memory performance independently. Because of high rates of these problems, the association of any defects in learning and memory in these patients cannot always be readily or definitively linked to PTSD, particularly without the use of more sophisticated tests known to dissociate among various etiologies.[28] In addition, tests such as the WMS(-R) are likely to lack sufficient difficulty for use with some PTSD patients who perform at a test's upper limits because of their higher functional status. Accordingly, such tests will fail to detect all but the most serious memory problems. In contrast, tests such as the CVLT, which are more challenging and contain measures of disparate memory abilities (e.g., retention over trials, primacy and recency, and effects of interference) are potentially more useful in PTSD.[29]

Still, without the testing of other higher cortical functions, it is difficult to assess if memory functions alone are affected in PTSD or if the performance of these patients reflects more generally disrupted cognitive abilities (e.g., frontally mediated processing inefficiencies). It is noteworthy that within some PTSD populations (e.g., combat veterans), high proportions of participants are likely to have learning disabilities or other developmental problems in their backgrounds[3,28,30] Such data raise speculation about whether earlier, predisposing characteristics constitute a vulnerability in this disorder for either the development of PTSD following trauma exposure or the manifestation of deficits in proximal domains (i.e., memory) when tested as adults.[31] As such, neuropsychologists and others might be well advised to assess a broader range of functions (including sequencing and organizational skills) which typically underlie the performance of memory tasks when evaluating PTSD in certain populations.[32] This approach would place memory assessment within a larger context and could help address whether memory systems per se are disrupted by this disorder or whether alterations represent more basic, structural, or systemic anomalies.

Generally, more definitive memory changes are found when experimental neuropsychological tests are employed with PTSD patients.[33,34] These tasks are typically more challenging in their attentional, memorial, and processing requirements; furthermore, their design permits the breaking apart of various memory components, enabling improved content analysis. To date, research findings have provided data in two important domains: (1) the nature of selective processing and retention in PTSD, and (2) the capacity for general recall under varying demand conditions.

Current findings suggest that individuals with PTSD in fact demonstrate changes in the processing of emotionally salient material[35] as well as in attention. These data confirm an enhanced attentional bias for trauma salient or trauma congruent stimuli. When complex processing tasks are employed, these alterations are likely to be seen in conjunction with markedly increased response latencies, suggesting the reallocation of attentional resources (as evidenced on Stroop tasks when emotionally laden words are employed[10,15,36]) and a pronounced susceptibility to trauma congruent interference. Whether these deficits will be confirmed on tasks involving nontraumatic stimuli is unclear at this time. However, it is essential that research be structured so that effects of stimulus content can be dissembled from basic processing and memorial requirements if correlates of memory in PTSD are to be elucidated.[37,38]

CURRENT EMPIRICAL FINDINGS

To summarize, findings of cognitive deficits in PTSD can be classified broadly in two categories: (1) those providing evidence for equivocal or no deficits across a range of functions (e.g., attention, verbal fluency, and visual tracking) and (2) those supporting memory deficits in verbal or visuospatial domains. A brief review of empirical research in each is provided.

Yehuda *et al.*[29] administered standard neuropsychological tests to individuals with and without PTSD and found that PTSD patients generally performed within normal limits on a variety of attention, immediate memory, and cumulative learning tasks. However, when tasks involved interference conditions, the performance of PTSD patients, specifically their capacity for retention, diminished. This finding preliminarily strengthens the argument that memory defects in PTSD may reflect underlying attentional disturbances. Evidence of other subtle cognitive changes comes from research involving the use of standard and experimental neuropsychological paradigms. Uddo *et al.*[39] examined the performance of male veterans with PTSD and found learning inefficiencies marked by decrements in cumulative learning across trials, a pattern not observed in individuals without this disorder. In addition, the performance of PTSD patients was characterized by more perseverative errors, problems in fluency and tracking, and sensitivity to proactive interference. Together, these deficits suggest the possibility of changes in brain mechanisms that support more complex and efficient learning.

Other evidence for minor cognitive and memory alterations comes from experiments on autobiographical memory in PTSD.[19,40] McNally *et al.*[19,40] found that PTSD patients tended to overgeneralize on tasks involving personal recall, suggesting limitations in their ability to distinguish effectively among various salient cues. Similarly, Schwarz *et al.*[41] demonstrated that individuals exposed to trauma were susceptible

to altering their retrospective reports of events, regardless of age. These changes included distortions in proximity to the event as well as in event sequencing. Furthermore, greater perceptual memory changes were associated with increasing levels of PTSD symptoms. However, these performance patterns were also associated with symptoms of anxiety and depression; hence, the specificity of this finding for PTSD is unclear.

Use of experimental laboratory tests of cognition offer a better opportunity to disentangle the components of memory performance in PTSD. To date, changes in recall have been found using both explicit (with conscious awareness) and implicit (without conscious awareness) memory paradigms. Because prior research on these memory systems suggests that they are distinct from each other, such findings might imply that memory problems in PTSD are diffuse or nonspecific, rather than selective.[42] However, closer examination of research findings shows that certain characteristics of memory tasks may play a role.

In terms of actual recall capabilities, data from explicit memory studies remain equivocal. Although Yehuda *et al.*[29] found that explicit memory in PTSD was intact, other studies have found evidence of impairment.[13] According to resource allocation models, one possible explanation is that explicit memory performance in PTSD is impacted by the diminution of attentional resources during processing conducted under conscious control. This theory, espoused by Siegel,[13] suggests that the emotional reactivity caused during trauma leads to cognitive disruption by dividing attentional resources and disturbing focal attention and effortful learning. Furthermore, only explicit memory is affected because material processed under less than full conscious awareness (i.e., implicit memory) is afforded greater scrutiny, especially when the content is emotionally salient. This model is predicated on the assumption that only conscious, effortful learning is disrupted within an integrated memory system.

Other data, which are not inherently contradictory, suggest that implicit memory remains intact or is potentially enhanced in PTSD. Using resource allocation or memory network models, this performance pattern could stem from an intensified focus on material that has prime informational importance, that is, data relating to conditions of threat or intense fear. Some research studies have hypothesized that this process is analogous to PTSD's reexperiencing symptom cluster, that is, behavioral manifestations of efforts to reintegrate unabsorbed cognitive and affective elements associated with the trauma.[43] In fact, several studies show that implicit memory performance in PTSD is likely either to (1) exceed that of normal control subjects or (2) be enhanced relative to PTSD patients' explicit memory abilities.[33,44] Importantly, these findings are most robust when threat consonant stimuli are employed, again suggesting the importance of conditioned associations. Together with evidence from autobiographical tasks, implicit memory data appear to confirm that the observed memory and learning alterations in PTSD stem more from affectively based effects of stimulus content rather than disruptions in basic memory systems per se.

Despite the early stage of neuroscientific research in PTSD, data identifying more selective memory problems offer some evidence for linkages between the pathophysiology of this disorder and memory. Sutker *et al.*[45] examined a pair of monozygotic adult twins and found that only the twin with PTSD showed deficits on tasks of nonverbal memory. New studies using brain imaging also offer preliminary evidence concerning brain change in PTSD and its relevance for cognitive status.

Bremner *et al.*[12] studied male Vietnam combat veterans with PTSD and found evidence for significant reduction in right hippocampal density (8%) compared to that of participants without this disorder. Furthermore, volumetric decrease was associated with concurrent deficits in *verbal,* short-term memory as measured by the WMS. Measures of other brain regions (e.g., temporal lobes) in these patients did not show similar decreases, suggesting the possibility that the hippocampus could play a distinctive role in the mediation of traumatic stress and its associated features.

Although these results are compelling, findings from other neuroanatomically based studies in PTSD suggest greater discretion. Stein[14] studied female survivors of childhood sexual abuse with and without PTSD and observed diminished right hippocampal density in abuse survivors as a group, irrespective of PTSD status. Thus, severe trauma exposure, rather than the ensuing PTSD, might be a determining factor. Furthermore, this study failed to demonstrate the presence of notable memory deficits in women either with or without PTSD, suggesting that hippocampal density was not directly implicated in the genesis of memory changes. Still, these studies reflect the complexity of content and methodological issues that will need to be addressed, including gender, age, intensity of trauma exposure, and symptom duration.

Data from studies in the neurosciences offer other evidence for contexts in which more localized memory deficits might occur in PTSD. Basic neurological research in patients undergoing temporal lobectomy, for example, has demonstrated the strong relationship between these brain regions and preserved functions of verbal and visual memory, substantiating the normal neural substrates for these functions.[46–49] Furthermore, Trenerry *et al.*[50] recently showed that destruction (i.e., removal) of portions of certain underlying subcortical areas (e.g., right hippocampus) produces similarly noteworthy declines in visual memory. However, most PTSD patients are not thought to have discrete brain lesions. Accordingly, evidence for the link between known structural damage and particular cognitive skills is far more substantial than that proposed for most psychiatric conditions.

CONCLUSION

Growing research suggests that certain cognitive alterations occur in PTSD. These changes are generally characterized by a preferential bias or increased attention associated with the presentation of threat-related stimuli, despite processing latencies. Accordingly, PTSD patients may evidence seemingly enhanced learning and retention of some trauma stimuli. In other instances, deficits in explicit recall are documented, but these are not uniformly observed. Importantly, the conditions under which explicit memory deficits occur are not well understood and are likely to reflect diverse factors such as baseline cognitive abilities, history of learning disabilities, and contributions of mood states.

Results of both enhanced and decreased memory functioning in PTSD raise the possibility of a bidirectional memory model. Such a model is consonant with the phenomenology of PTSD which reflects alternating elements of phasic sensitization/hyperreactivity and avoidance/numbing. As brain and behavioral mechanisms of PTSD are better understood, complex interactions between intrinsic cortical and external or contextual factors should help to elucidate the conditions under which

various cognitive mechanisms operate. Still, certain methodological issues warrant mention. First, future research should address the specificity of any observed memory changes for this disorder. To date, studies have relied nearly exclusively on contrasts between PTSD and well-adjusted samples, precluding critical diagnostic comparisons. Subsequent studies will need to employ a range of appropriate, matched psychiatric comparison groups to demonstrate the ways in which deficits are specific to PTSD and not generic to psychiatric disturbance overall[51] or subsets of anxiety and affective disorders in particular. Relatedly, study designs should more carefully assess extant comorbidities in PTSD patients and control groups, especially given the high rates of comorbid disorders in PTSD and the demonstrated impact of such disorders (e.g., major depression) on memory performance.

Second, improved assessment of baseline cognitive abilities in respondents is strongly recommended. This should be conducted across the range of cortical functions, so that evaluation of learning and memory skills can be considered in the appropriate context and at various levels of difficulty. Special attention should be paid to the possibility of early developmental anomalies or learning disabilities given the growing interest in family genetics and inborn vulnerabilities for conditions such as PTSD.[31,52] Third, the performance of PTSD patients on neuropsychological measures to date indicates that more sophisticated tests of memory and attention are needed. Currently, interpretation of some study results is constrained by potential ceiling effects of certain tests, limiting the ability to detect subtle performance alterations. Also, both standard and experimental neuropsychological tests should be employed that permit closer examination of critical components of learning and memory, for example, response latencies, sequential processing, span of apprehension, and susceptibility to various forms of interference. Finally, we encourage the development of prospective studies in trauma-exposed young individuals with and without the PTSD diagnosis, as this research offers the opportunity to examine contributions of age, developmental status, plasticity, and adaptational mechanisms to cognitive performance across the spectrum of stress responses.

SUMMARY

Mental health professionals have employed a variety of clinical and experimental neuropsychological tests for exploring purported memory alterations in PTSD. Protocols range from standard tests of immediate and delayed learning, recall, and recognition to elaborate paradigms using experimental stimuli for assessment of information-processing skills. Whereas the former have typically focused on general learning and memory capabilities, experimental paradigms have examined the role of trauma-related cues and their impact on remembering. Findings to date suggest that memory abilities in PTSD patients range from intact to mildly impaired on general tests of verbal or visual memory. At the same time, memory tests involving trauma-specific stimuli point to alterations in cognitive information processing, specifically, an attentional bias manifested by changes in speed, accuracy, and depth of processing. The role of a semantic information network involving enhanced specificity for trauma cues is discussed along with possible implications for brain structures and theories of PTSD.

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